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Clinical Note

Abraham Lincoln and Aortic Insufficiency

The Declining Health of the President

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ACCORDING TO MANY OBSERVERS and subsequent scholars, the health of Abraham Lincoln in his last year of life was such as to preclude completion of his second term of office even if he had not been assassinated. Understandably, in the absence of a known organic basis for that unfavorable prognosis, the decline in the physical well-being of the President has been attributed to emotional factors attendant upon the burdens of office and a tragic war—an impression that only positive evidence, the subject of this report, can perhaps obviate.

Background and Hypothesis

Several years ago a case of the Marfan syndrome in a young boy under my care was presented and a pedigree given indicating his descent from Mordecai Lincoln II, a great-great grandfather of Abraham Lincoln.[1] Descriptive and genetic evidence was also submitted demonstrating that the striking morphologic characteristics of the 16th president were likewise those of the Marfan syndrome as derived from the ancestor held in common with the patient reported. While in the original communication only involvement of the skeletal and visual apparatus was documented for the President, material by which to infer in his case a cardiovascular lesion typical of arachnodactyly has long been available. Before this observation was reported, however, it was felt desirable, because of the indirect nature of the data, to find a way to make this impression objective, and only recently has a method of accomplishing this been found. Evidence is therefore now offered suggesting that President Lincoln, two years before his assassination, had physical findings consistent with aortic insufficiency, as a complication of the Marfan syndrome.

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The Evidence

The evidence suggesting a cardiovascular lesion in Abraham Lincoln actually evolves from certain observations and remarks of the president himself, together with those of Noah Brooks, a newspaperman, as they reviewed a photograph for which Mr. Lincoln had posed a short time previously[2,3] (Figure 1). In the episode, as reported by Hamilton and Ostendorf,[2] the president was speaking:

> "I can understand why that foot should be so enormous," he said to Brooks—"It's a big foot anyway, and it is near the focus of the instrument. But why is the outline of it so indistinct and blurred? I am confident I did not move it."
>
> Brooks suggested that the throbbing of the arteries may have caused an imperceptible motion.
>
> The President crossed his legs and watched his foot. "That's it! That's it!" he exclaimed. "Now that's very curious, isn't it?"[3]

Aortic insufficiency (AI) being a common lesion in the Marfan syndrome, the clinician will recognize in the suspended throbbing and pul-

Figure 1.—The Photo-Kymographic Evidence: Lincoln noted that the outline of his left boot was blurred, and Brooks expounded upon the theory that the throbbing arteries caused the leg to move almost imperceptibly.[3]

sating foot of the President, a phenomenon entirely compatible with the hemodynamic effects of aortic regurgitation.

Fundamentally, such visible pulsations result from sudden filling of the large collapsed arteries accompanying aortic regurgitation. Corrigan in 1832 described this effect in the carotid, temporal and other like vessels while similar pulsations have since been described in the uvula, the liver and the capillaries. Even more applicable, cardiologist Luisada emphasizes, as a manifestation of the "vascular dance" accompanying AI, the abrupt "rhythmic extension of one leg when crossed over the other coincident with systole."[4] Further, the eminent Sir Thomas Lewis states specifically that the jerking water-hammer quality of the pulse-wave of aortic incompetence "is extreme in the dorsum of the foot."[5]

Other studies reveal that in AI, the blood pressure in the femoral artery is greatly exaggerated over that of the brachial and since the femoral artery is in a direct line with the aortic stream while the brachial issues from the aorta at a right angle, the femoral receives both the pressure and the very considerable velocity head of the aortic stream. This increased pressure and velocity head is precisely the mechanism that may produce pulsatile motion in a suspended leg and foot in AI, a lesion that in the Marfan syndrome is the direct result pathologically of dilatation of the aortic ring or myxomatous change of the aortic valve.

Independent objectifying evidence supporting the plausibility of the hypothesis offered is available in an analogous situation recently documented by a young journalist who himself had the Marfan syndrome. This man, in a recent book describing his personal medical experiences with ectopia lentis, loose-jointedness and aortic insufficiency, noted most perceptively:

> "My heart had its work cut out . . . There was no need to feel my pulse. Its heavings were all too readily discernible in my neck, at my wrist and even on the back of my hand. And when I crossed my legs and let my muscles relax, the upper leg jumped up and down like a spring, keeping perfect rhythm with the beat of my heart muscle."[6]

That a non-physician with the same condition that affected Abraham Lincoln made this observation in himself, would appear to lend considerable reliability and validity to the similar observation and analysis made a hundred years previously by the laymen Lincoln and Brooks.

The pulsating suspended leg and foot phenomenon, which may be called the Lincoln-Brooks sign of aortic insufficiency, is made possible by the hinge-like arrangement of the related body parts. From this arises the possibility that laxness with hypertensibility of the joints, as commonly seen in arachnodactyly, may facilitate pulsatile motion of a suspended extremity even in the presence of lesser degrees of regurgitation than otherwise required for this phenomenon in cases of aortic insufficiency without hyperextensibility. While Lincoln was many times described by his contemporaries as "loose-jointed," further evidence specifically for the laxness of his ankle joint which may have made his foot, in addition to his leg, more responsive to minor degrees of regurgitation, is found in a description by Lincoln's law associate William Henry Herndon: "In walking, Mr. Lincoln put the whole foot flat down on the ground at once, not landing on the heel. He lifted his foot all at once—not lifting himself from the toes and hence had no spring or snap . . . to his . . . walk."[7]

While undoubtedly a mere coincidence, the fact remains that the pose assumed for the photograph under consideration, had Lincoln's lower extremities crossed, with the overhanging foot close to the lens where its arc, increased first by the rhythmically extended leg and then by the still oncoming steep pulse-wave reaching the foot, was optimal for producing "photokymographic" documentation of the President's "altered" circulatory dynamics.

As to other findings in aortic insufficiency, no blood pressure apparatus was clinically available in Lincoln's time by which to establish that there was a wide pulse-pressure. Regarding also the usual aortic diastolic decrescendo murmer: it may in any given case be absent or extremely difficult to elicit or localize, particularly in the presence of an altered configuration of the chest. Significantly, Mr. Lincoln has been shown[1] to have, as a skeletal component of the Marfan syndrome, a pectus excavatum which shifts the heart well into the left hemithorax, thereby rendering many of the cardiac findings less than typical. Furthermore, when detected, this murmur reflects diagnostically only the same pathologic condition that produces a bounding pulse, and in that sense the murmur is only one of many cardiovascular signs of aortic incompetence. The absence therefore of evidence for a diastolic aortic murmur in the President does not detract from the present diagnostic impression.

Discussion

Considering, then, the presence of the Marfan syndrome as previously established in Mr. Lincoln; the frequency of aortic valvular incompetency in that condition; the hemo-dynamics of aortic regurgitation; the graphic data of the Gardner photograph as detected by the President himself; the hypothesis of Brooks and its clinical confirmation by Lincoln; the leg and foot signs in the medical literature; the analogous clinical findings by a non-physician with arachnodactyly and the diagnostic criteria quoted, the evidence in the case of the President becomes highly consistent with aortic insufficiency and regurgitation.

Adding a cardiovascular lesion consistent with arachnodactyly to the previously documented skeletal and visual findings in the case of the President reinforces considerably the original diagnosis of the Marfan syndrome as already authoritatively accepted elsewhere[8] by completing involvement in all three major body systems any one or more of which may participate in the total spectrum of arachnodactyly as a heritable disorder of connective tissue.

Further, from the perspective of the present diagnostic impression, the course of the declining health of the President can be shown to be consistent with the late slow form of cardiac decompensation highly characteristic of AI. In relation to this, the period preceding Mr. Lincoln's violent death was one of easing burdens, lessening anxiety and lightening of spirit as the War Between the States drew to its long-awaited close. The health of the President, however, continued to deteriorate ever more rapidly, a paradox inconsistent with psychic stress as the one responsible factor. More compatible with the physical decline of Mr. Lincoln is a progressive decrease in cardiac reserve subsequent to long-standing aortic insufficiency, the course of which was abruptly terminated by his assassination on April 14, 1865.

In conclusion, if the diagnosis of the Marfan syndrome has resolved to any extent some of the enigma long surrounding President Lincoln, it also highlights one of the foremost problems of the genetic era—genetic control. As expressed by one authority citing by way of example the Marfan syndrome and Abraham Lincoln: ". . . who would dare wish him eugenically suppressed?"[9]

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